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CASE REPORT | LIVER

A Fatal Case of Hepatic Portal Venous Gas Associated With Hemodialysis

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ABSTRACT

Hepatic portal venous gas is a rare cause of acute abdomen caused by leakage of air from the gastrointestinal tract to the portal venous system. The mortality is high, particularly when associated with intestinal ischemia or necrosis. We describe a fatal case of hepatic portal venous gas and pneumatosis intestinalis due to hemodialysis-related hypotension and severe atherosclerotic disease.

INTRODUCTION

Air in the liver due to presence of gas in the portal venous system is a rare clinical scenario. Air enters into the portal venous system from the gastrointestinal tract through a mechanical breach due either to direct injury to the intestinal mucosa or a gas-producing organism present in the lumen. For elderly patients who already have significant atherosclerotic disease involving the mesenteric vasculatures, a second incident oftransient hypotension of any cause may precipitate intestinal ischemia leading to hepatic portal venous gas (HPVG) entry. Meticulous monitoring of hemodynamic status during any medical procedure and quick resuscitation may prevent the onset of such a fatal condition.

CASE REPORT

A 78-year-old man presented with diffuse abdominal pain that had started as cramping mid-abdominal pain 12 hours priorduring his regular hemodialysis session. He had a past medical history of Type 2 diabetes mellitus, end-stage renal disease, hypertension, peripheral vascular disease, and sick sinus syndrome. He had also vomited non-bilious, non-bloody, comprising stomach contents several times. Initial blood pressure was 102/54 mm Hg, pulse rate was 115 beats/min, breath rate was 29 breaths/min, and oxygen saturation was 89% on room air. He appeared agitated and rated the pain intensity at 10 of 10. His abdomen was firm, tender, distended, and tympanitic, with guarding and rebound tenderness. Bowel sounds were absent in all quadrants.

Initial investigations showed a white blood cell count of 15,500 cells/μL with 12% of bands, and an absolute neutrophil count of 12,100 cells/μL. Hemoglobin was 13.1 g/dL and hematocrit was 38.7%. Liver function tests and amylase and lipase levels were within the normal limit. Lactic acid was 6 mmol/L. Plain abdominal x-ray suggested ileus, and ultrasound showed a heterogeneous appearance to the liver. Abdominal/pelvic computed tomography (CT) with contrast showed a large amount of gas in the hepatic portal vein and branches (Figure 1). Air was also present in the superior mesenteric vein (Figure 2). CT also revealed pneumatosis intestinalis of the small bowel with diffuse small bowel wall thickening, likely due to ischemia (Figure 3). There was severe atherosclerotic disease of the celiac axis and the superior mesenteric artery, as well as of the aorta and the remainder of its major branches (Figure 3).

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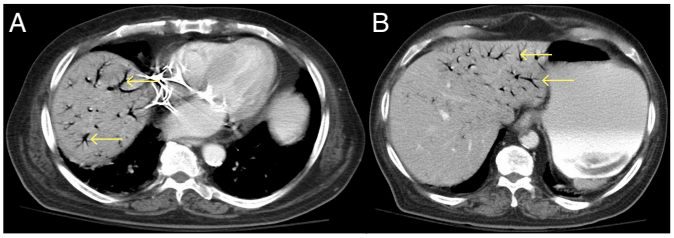

Figure 1. Abdominal CT showing (A) air within the branches of hepatic vein (arrows) and (B) air in the hepatic venous system extended within 2 cm of the liver capsule (arrows).

The patient was started on aggressive intravenous hydration with ciprofloxacin and metronidazole. Emergent laparotomy was considered, but given his age and comorbid conditions, we decided on a conservative approach. After discussion with the patient's family, he was transferred to the inpatient hospice service where he died within 24 hours of initial presentation.

DISCUSSION

HPVG is a rare clinical presentation acute abdomen. It was first reported as a case of infantile necrotizing enterocolitis in 1955.[1] In a review of 64 cases, mortality was reported at approximately 75%.[2] A later cause-specific analysis from a review of 182 cases revealed an overall mortality of 39%, with bowel necrosis as the main precipitating event in most of cases.[3] The occurrence of the HPVG is most frequently caused by mechanical injury to the bowel lumen or by gas-producing bacteria in the intestine.[4] Conditions such as inflammatory bowel disease, ischemia, or increased lumen pressure may damage bowel wall integrity and allow the dissection of gas into the intestinal wall and portal system. We hypothesize that our patient developed intestinal ischemia due to hypoperfusion of the mesenteric vessels related to severe atherosclerotic disease, aggravated by a hypotensive episode during hemodialysis.

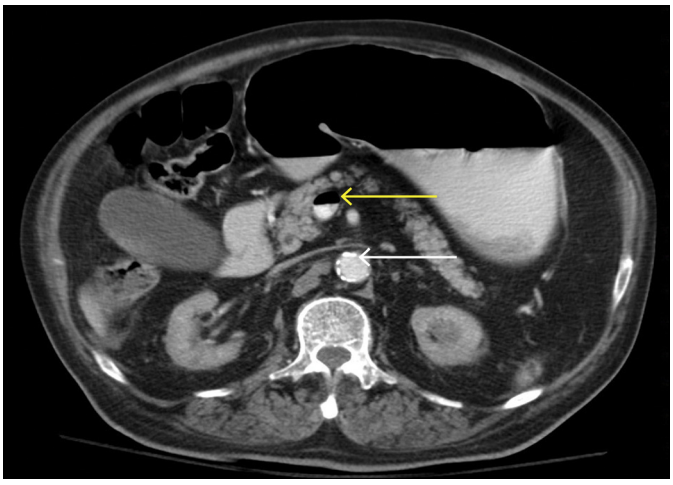

Figure 2. Abdominal CT showing air in the superior mesenteric vein (yellow arrow) and atherosclerosis of the aorta (white arrow).

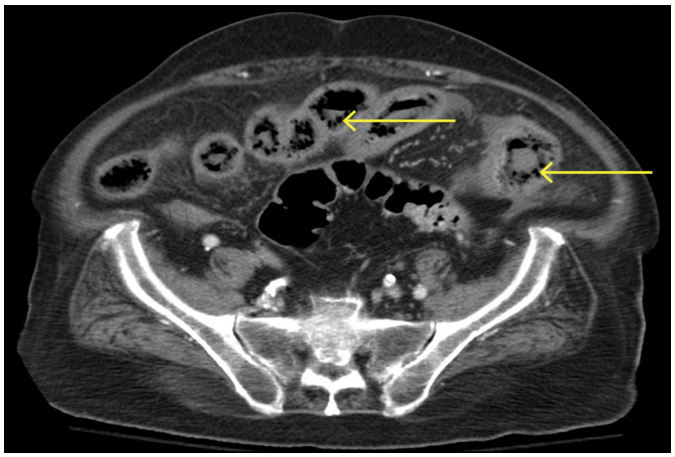

Figure 3. Abdominal CT showing air within the wall of the small intestine (arrow).

It is important to differentiate between HPVG and pneumobilia, which is gas in the hepatobiliary tree. In HPVG, air will extend within 2 cm of the liver capsule. Gas will move peripherally due to centrifugal flow of blood.[5] In pneumobilia, air in the biliary tract remains central tothe portal hilum and does not extend to within 2 cm of the liver capsule.[5] Gas moves in the biliary tree toward the porta hepatis because of the centripetal force of bile.

Pneumatosis intestinalis is a radiological sign commonly associated with HPVG. It occurs due to translocation of luminal gas into the bowel mucosa forming small air pockets. The presence of HPVG is associated with Transmural infarctions and mortality occur more frequently in the presence of HPVG than in cases of isolated pneumatosis intestinalis.[6] Because the mortality rate depends greatly on whether HPVG is associated with bowel necrosis, early diagnosis is essential to initiate emergent treatment. One study of HPVG patients found that low systolic blood pressure (<108 mm Hg), elevated LDH cholesterol (>387 U/L), and presence of intestinal pneumatosis were independent risk factors for necrotic bowel.[4] Patients with all 3 criteria had 100% sensitivity and 78.9% specificity for bowel necrosis. Another study found that older age (60 years), peritoneal signs, and elevated BUN (>25 mg/dL) were all associated with ischemia or necrosis in patients with HPVG. CT is the best modality for the diagnosis of HPVG, which appears as multiple branching radiolucencies extending up close to the liver capsule. Ultrasound and plain radiography have been used but are less sensitive.

Treatment for HPVG is based on the underlying cause of the disease entity. More benign conditions are generally

managed conservatively with broad-spectrum antibiotics, volume resuscitation, and bowel rest. Cases with signs of ischemia, necrosis, and sepsis are likely to require surgical intervention. The rate of survival of surgical and conservative management group are 73% and 60%, respectively.[7]

DISCLOSURES

Author contributions: T. Begum wrote the article and analyzed the data. M. Ahmed wrote and critically revised the manuscript, and is the article guarantor.

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Informed consent was obtained for this case report.

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